

Segregation in a competing and evolving population

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We study a recently proposed model in which an odd number of agents are competing to be in the minority. In the model, the agents have one strategy in hand which is to follow the most recent history. Each agent is also assigned a value p , which is the probability that an agent will follow the trend. Evolution is introduced through the modification of the value of p when the performance of an agent becomes unsatisfactory. We present numerical results for the distribution of p values in the population as well as the average duration between modifications at a given p for different values of the parameters in the model. Agents who either always follow the trend or always act opposite to the trend, tend to out-perform the cautious agents. We also point out the difference between the present model and a slightly modified model in which a strategy is randomly assigned to every agent initially.

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I. INTRODUCTION

Agent-based models of complex adaptive systems (CAS) provide invaluable insight into the highly non-trivial global behaviour of a population of competing agents [1]. Typically, these models involve agents with similar capability competing for a limited resource. The agents share the same global information, which is in turn generated by the behaviour of the agents themselves, and they learn from past experience. A realistic situation, for example, is where the index of a stock market is made known to every participating agent: the agents must then decide whether to buy or sell based on this global information. It is, therefore, not surprising to see that these models constitute an active part of the growing field of econophysics [2,3].

One of the earliest models was proposed by Arthur [4]. The model, which is referred to as the bar-attendance model, consists of N agents trying to decide whether to attend a bar with a seating capacity L ($L < N$). The attendance in the past weeks is announced to all agents, forming the global information. A correct decision is to attend (not to attend) the bar with an attendance less than or equal to (higher than) the cutoff L . It turns

out that [4,5] the population cooperates or self-organizes in the sense that the attendance in each turn is usually close to L and the variance of the attendance can show a minimum with increasing adaptability of the individual agents. This model shows a few features typical of CAS. The cooperation in the system necessarily requires an inhomogeneous population. If all agents were to decide according to the same strategy, all of them would act identically and hence lose. In fact, there is no *a priori* best strategy and a strategy good at one point in time will become bad when too many agents use it. The agents hence interact through the creation and sharing of the global information, and are forced to make decisions based on inductive, rather than deductive thinking [6].

The bar-attendance model is rather complicated in that the actual attendance is announced. Challet and Zhang proposed a binary game, called the minority game, in which an odd number N of agents are competing to be in the minority group [7,8]. The agents decide to go into one of two rooms, with the winners being those in the room with fewer agents. The outcomes are announced and form the global information. The agents are assumed to have limited and similar capabilities in that they all decide based on the outcomes of the recent m turns. There are a total of 2^m possible history bit-strings of length m , thus forming a strategy space consisting of a total of 2^{2^m} strategies. Each agent picks s strategies from this pool initially and uses the one with the best accumulative performance in deciding the next move. Detailed numerical calculations have revealed that the standard deviation (SD) in the attendance in a room shows a minimum as a function of m at a value corresponding to $N \cdot s \sim 2 \cdot 2^m$. Johnson and coworkers [9] suggested that the features can be understood in terms of the dynamical formation of crowds consisting of agents using, say, the best strategy at a particular moment in time and anticrowds consisting of agents using the strategy anti-correlated to the crowd. Basically the degree of overlap in the strategies among the agents plays an important role. The condition $N \cdot s \sim 2 \cdot 2^m$ favours the formation of crowds and anti-crowds of comparable size and leads to a minimum in the SD. The idea can be formulated quantitatively in terms of the Hamming distance [10] between strategies.

In both the bar-attendance and the basic minority game, the strategies, once distributed at the beginning of the game, are fixed and the agents adapt by choosing the best-performing strategy in hand. Recently, we

proposed a simple model consisting of an *evolving* population in which the agents can adapt by changing their behaviour without the limitation imposed by the strategies being initially distributed [11,12]. In this paper, we report results on this model focusing on the possible segregation of the population as a result of competition and evolution. We introduce the model in Sec.2. Results are presented in Sec.3. In Sec.4, we summarize our results and point out the difference between our model and a similar model proposed in Ref. [10] in an attempt to formulate a theory of the present model.

II. THE GENETIC MODEL

We introduce a simple, yet realistic, model for an evolving population containing adaptive agents who compete to be in the minority. Inspired by Ref. [7], we consider the model of an odd number N of agents repeatedly choosing to be in room “0” or room “1”. After each agent has independently chosen a room, the winners are those in the minority room. The “output” for each time step is a single binary digit denoting the minority room. Each agent is given the information of the most recent m outcomes. Each agent also has access to a common register or “memory” containing the outcomes from the most recent occurrences of all 2^m possible bit strings of length m . Consider, for example, $m = 3$ and denote $(xyz)w$ as the $m = 3$ bit string (xyz) and outcome w . An example memory would comprise (000)1, (001)0, (010)0, (011)1, (100)0, (101)1, (110)0, (111)1. Following a run of three wins for room “0” in the recent past, the winning room was subsequently “1”. Faced with a given bit string of length m , it seems reasonable for an agent to simply predict the same outcome as that registered in the memory. The agent will hence choose room “1” following the next 000 sequence. If “0” turns out to be the winning room, the entry (000)1 in the memory is then updated to be (000)0. Simply put, each agent looks into the most recent history for the same pattern of m bit string and predicts the outcome using the history. In effect, each agent holds one strategy and all agents hold the same strategy, with the strategy being *dynamical*. The strategy is hence to follow the trend. However, if all N agents act in the same way, they will all lose. A successful agent is one who can follow a trend as long as it is valid and to correctly predict when it will end. To incorporate this factor into our model, we assign to each agent a single number p , which we refer to as the “gene”-value. Following a given m -bit sequence, p is the probability that the agent will choose the same outcome as that stored in the memory, i.e., he will follow the current predictor. An agent will reject the prediction and choose the opposite action with probability $1 - p$.

To incorporate evolution into our model, we assign +1 (−1) point to every agent in the minority (majority) room at each time step. If an agent’s score falls below

a value d ($d < 0$), his gene value is modified. The new p value is chosen randomly from a range of values centered on the old p with a width equal to R . We impose reflective boundary condition to ensure that $0 \leq p \leq 1$. Our conclusions do not depend on the particular choice of boundary conditions. For $R = 0$, the agents will not change their gene values at all. For $R = 2$, the new gene value is uncorrelated with the old one upon modification.

III. RESULTS

We have carried out detailed numerical studies of our model. Initially, each agent is randomly assigned a gene value in the range $0 \leq p \leq 1$. The population is allowed to evolve. We focus on two quantities, $P(p)$ and $L(p)$, in the asymptotic limit. Here $P(p)$ is the frequency distribution of gene values, typically taken in the long time limit over a time window and normalized to unity; $L(p)$ is the lifespan defined as the average length of time a gene value p survives between modifications. Figure 1 shows $L(p)$ and $P(p)$ (inset) as a function of p for different values of m . The other parameters are taken to be $N = 101$, $R = 0.2$ and $d = -4$. The most interesting feature is that $P(p)$ becomes peaked around $p = 0$ and $p = 1$, with a similar behaviour in $L(p)$. The results are insensitive to the initial distribution of p . Surprisingly the results indicate that agents who either always follow or never follow what happened last time, generally perform better than cautious agents using an intermediate value of p . Figure 1 also shows that there is no explicit dependence on m for $P(p)$ and $L(p)$. We have also checked that different values of d do not change the normalized distribution $P(p)$. The lifespan $L(p)$ obviously does depend on d as shown in Fig. 2 for $d = -1, -2, \dots, -9$. A more negative value of d leads to a longer time between modifications and hence a longer time in approaching the asymptotic $P(p)$. The inset in Fig. 2 shows $L(p)/|d|$ as a function of p for different values of d and all the data fall onto one curve showing $L(p) \sim |d|$.

Figure 3 shows the dependence of $L(p)$ and $P(p)$ (inset) as a function of p for different values of R . Different values of R affect the time it takes to approach the asymptotic limit, since a larger value of R gives the agent a larger jump in gene-value space in order to arrive at the final $P(p)$. However, $L(p)$ and $P(p)$ do not depend on the value of R . It should be pointed out that even when $P(p)$, and hence $L(p)$, takes on its asymptotic form, agents are still constantly modifying their gene values. It is a dynamical state in that agents are changing their gene values while keeping the form of $P(p)$ unchanged. Figure 4 shows $P(p)$ and $L(p)$ for games with different numbers of agents. The normalized $P(p)$, again, does not depend on N , while the $L(p)$ is generally higher for games with larger N . Our results thus show that the segregation in the population as indicated in $P(p)$ is robust and insensitive to the choice of parameters in our model. It merely

comes from the desire of the agents to do the opposite of the majority. The segregation implies that the population as a whole samples the microstates of the system, i.e. the different possible distributions of the gene value in the population, unevenly as time evolves. There are microstates in which the total points deducted per turn are relatively small and the population tends to stay in these microstates longer giving rise to the segregation [11].

Note also that the results are symmetrical about $p = 1/2$ for both $P(p)$ and $L(p)$. Basically, it follows from a symmetry in the game in that the past behaviour contains the same information for every agent, and hence there is no advantage to any agent. Therefore, the game has the same distribution $P(p)$ regardless of whether the real, or static, or random history is followed; and we could replace the strategy at any moment by its inverse. Thus, agents with gene values p and $1 - p$ are doing equally well.

IV. SUMMARY

We have presented numerical results for our genetic model consisting of an evolving and competing population. Agents who always follow or always act opposite to the predictor, out-perform the cautious agents. Recently, an attempt was made to explain quantitatively the form of the asymptotic gene distribution $P(p)$ [10]. The theory, while succeeding in obtaining a $P(p)$ similar to those reported here, was formulated based on a slightly different model. In the modified model, one strategy in the pool of 2^{2^m} possible strategies (corresponding to a game with memory m) is assigned randomly to each player at the beginning of the game, in addition to the gene value. Thus, the modified model in Ref. [10] corresponds to a minority game with $s = 1$ plus the inclusion of a gene value p for each agent. Although the modified model and the present model both correspond to cases with one strategy per agent, the strategy is dynamical in the present model and is constantly updated; in the modified model the strategy is fixed. It is, therefore, interesting to check numerically if the two models give identical results for $P(p)$. Figure 5 shows $P(p)$ for the modified model with $m = 1, 2, \dots, 10$. We notice that, unlike our present model, the modified model's $P(p)$ depends on m with the small m limit approaching the result of our model. The difference between the two models comes from the fact that for large values of m , the strategies held by the agents in the modified model are likely to be uncorrelated. In this case, each agent cannot adapt to the behaviour of the other agents and the distribution $P(p)$ becomes flatter as m increases. Interestingly, the quantitative analysis in Ref. [10], which is based on the modified model, does not give an m -dependent $P(p)$.

A more complete theory of our present genetic model can be formulated by investigating the attendance dis-

tribution in one of the two rooms. The approach [13] is to relate the average success rate of an agent in an N -agent game to a $(N - 1)$ -agent game with a particular agent being singled out, and to derive an expression for the average success rate in which the effect of the complicated interaction (and self-interaction) of the agents is isolated. Such a consideration leads to an average success rate $\tau(p)$ for an agent using a gene value p of the form $\tau(p) \sim 1/2 - A(N)p(1 - p)$, where A is an N -dependent parameter which decreases with N . The lifespan $L(p)$ is related to the average success rate by $L(p) \sim d/(1/2 - \tau(p))$ [10], hence leading to the symmetry about $p = 1/2$ for both $L(p)$ and $P(p)$ as discussed in the last section [14]. Results along this line will be reported elsewhere [13].

Our model forms the basis for incorporating various interesting complications. One possibility is that the agents, instead of competing to be in the minority group, are trying to attend a room with a specific cutoff capacity L ; the winners are those deciding to attend (not to attend) with the turnout being less than or equal to (greater than) the cutoff capacity. In this case, it is possible for the population distribution $P(p)$ to become frozen, i.e. no further modifications of gene values among the agents, as time evolves for large (or small) enough value of the cutoff [15].

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- [1] J.H. Holland, *Emergence: From chaos to order*, (1998) (Addison-Wesley, Reading); *Hidden Order: How adaptation builds complexity* (1995) (Addison-Wesley, Reading).
 - [2] H.E. Stanley, Computing in Science & Engineering Jan/Feb (1999) 76; Physica A **269** (1999) 156.
 - [3] See the proceedings of the International Workshop on Econophysics and Statistical Finance published in Physica A **269** (1999) 1-183.
 - [4] W.B. Arthur, Amer. Econ. Rev. **84** (1994) 406; Science **284** (1999) 107.
 - [5] N.F. Johnson, S. Jarvis, R. Jonson, P. Cheung, Y.R. Kwong and P.M. Hui, Physica A **258** (1998) 230.
 - [6] See, for example, the collection of articles in the special issue entitled "Beyond Reductionism" in Science **284** (1999) 80 - 109.
 - [7] D. Challet and Y.C. Zhang, Physica A **246** (1997) 407; *ibid.* **256** (1998) 514; *ibid.* **269** (1999) 30; D. Challet and M. Marsili, cond-mat/9904071, 9904392, 9908480.
 - [8] R. Savit, R. Manuca and R. Riolo, Phys. Rev. Lett. **82** (1999) 2203.
 - [9] N.F. Johnson, M. Hart and P.M. Hui, Physica A **269** (1999) 1.

- [10] R. D'Hulst and G.J. Rodgers, Physica A **270** (1999) 514; cond-mat/9908481.
- [11] N.F. Johnson, P.M. Hui, R. Jonson and T.S. Lo, Phys. Rev. Lett. **82** (1999) 3360.
- [12] N.F. Johnson, P.M. Hui and T.S. Lo, Phil. Trans. Royal Soc. London A **357** (1999) 2013.
- [13] T.S. Lo, P.M. Hui and N.F. Johnson (unpublished).
- [14] L.-H. Tang, private communication. The author has argued that $L(p) \sim 1/p(1-p)$ using a random walk argument.
- [15] N.F. Johnson, D.J.T. Leonard, P.M. Hui and T.S. Lo, cond-mat/9905039.

FIG. 1. The lifespan $L(p)$, which is the average duration between modifications for a gene value p , as a function of gene value p for $m = 1, 2, \dots, 8$. The inset shows the distribution of gene values $P(p)$ as a function of p for different values of m . Both $L(p)$ and $P(p)$ are insensitive to m . The other parameters are $N = 101$, $d = -4$ and $R = 0.2$.

FIG. 2. The lifespan $L(p)$ for different values of d . The curves at $p = 0.5$ from bottom to top correspond to $d = -1, -2, \dots, -9$. The inset shows that all the data fall onto one curve if we plot $L(p)/(-d)$ as a function of p . The other parameters are $N = 101$, $m = 3$ and $R = 0.2$.

FIG. 3. The lifespan $L(p)$ and the distribution of gene values $P(p)$ (inset) as a function of p for $R = 0.1, 0.2, \dots, 2.0$. Note that both $L(p)$ and $P(p)$ are insensitive to R . The other parameters are $N = 101$, $m = 3$ and $d = -4$.

FIG. 4. The lifespan $L(p)$ as a function of p for $N = 11, 21, \dots, 81$. The inset shows $P(p)$ for different values of N . The other parameters are $m = 3$, $d = -4$ and $R = 0.2$.

FIG. 5. The distribution of gene values $P(p)$ as a function of p for the modified model in which each agent is assigned a strategy initially. At $p = 0.5$, the curves from bottom to top correspond to $m = 1, 2, \dots, 10$. The $P(p)$ depends on m in the modified model in contrast to the genetic model.









